Product Datasheet

BYL719

Catalog No. **BP-02122** · CAS **1217486-61-7** · Form **free base** · Physical state **solid** · Color **Off-white to light yellow** · Version **1.0** · Revision **2026-09-02**

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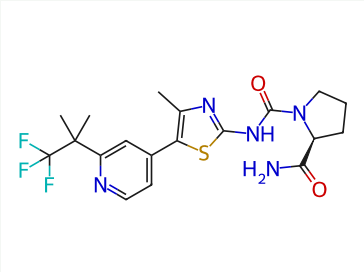
Target	Research area
PI3Kα	**Infection; Neurological Disease; Metabolic Disease; Cancer**

Molecular formula	$C_{19}H_{22}F_3N_5O_2S$
Molecular weight	**441.4700**

STRUCTURE



DESCRIPTION

Alpelisib (BYL719) is a potent and selective PI3Kα inhibitor with IC50 of 5 nM in a cell-free assay, and minimal effect on PI3Kβ/γ/δ. Phase 2.

BIOLOGICAL ACTIVITY

In Vitro

Alpelisib (0-5 μM; 14 d) significantly inhibits the clonal growth of MCF-7 and T47D breast cancer cells in 2D colony formation assays[1].

Alpelisib (5 μM; 5 d) reduces the mammosphere formation efficiency of MCF-7 and T47D breast cancer stem cell-like (BCSC-like) cells in a dose-dependent manner[1].

Alpelisib (0-10 μM; 10 d) significantly reduces the spheroid diameter of MCF-7 and T47D breast cancer stem cell-like (BCSC-like) cells in 3D culture systems, and inhibits their stem cell properties and drug resistance[1].

Alpelisib (1 μM; 24 h) significantly reduces the protein levels of the stem cell markers Nanog, Sox2, and OCT3/4 in MCF-7 and T47D breast cancer stem-like cells (BCSC-like cells)[1].

Alpelisib (10 μM; 10 d) inhibits adipogenesis, thereby attenuating adipocyte differentiation of primary LipPD1 lipoma cells in 2D culture systems and reducing the volume of 3D LipPD1 lipospheres[2].

Combination treatment with Alpelisib (10 μM; 4 d) and a 1 μM SGK3 inhibitor (VPS34-IN1 (HY-12795) or SGK3-IN) produces enhanced antiproliferative activity in Alpelisib GMP-resistant MCF7R and T47DR breast cancer cells, with a synergistic effect observed for the Alpelisib+SGK3-IN combination[3].

In Vivo

Alpelisib (50 mg/kg; i.p.; daily) exerts partial antitumor activity against alpelisib-resistant MCF7R breast cancer xenografts, with maximum efficacy achieved when combined with SGK3 knockdown[3].

Alpelisib (50 mg/kg; i.p.; daily) exerts partial antitumor activity against alpelisib-resistant T47DR breast cancer xenografts, with maximum efficacy achieved when combined with SGK3 knockdown[3].

Alpelisib (25 mg/kg; p.o.; daily; 4 weeks) significantly inhibits tumor growth, reduces tumor cell proliferation, and increases tumor cell apoptosis in a PIK3CA-mutant gastric cancer xenograft model with 100% survival of treated mice over 4 weeks[4].

SOLUBILITY

Soluble in DMSO

REFERENCES

[1]. Yu L, et al. Effects of BYL-719 (alpelisib) on human breast cancer stem cells to overcome drug resistance in human breast cancer. Front Pharmacol. 2024;15:1443422. Published 2024 Oct 14.

[2]. Kirstein AS, et al. The Novel Phosphatidylinositol-3-Kinase (PI3K) Inhibitor Alpelisib Effectively Inhibits Growth of PTEN-Haploinsufficient Lipoma Cells. Cancers (Basel). 2019;11(10):1586. Published 2019 Oct 17.

[3]. Kang T, et al. The SGK3/GSK3β/β-catenin signaling promotes breast cancer stemness and confers resistance to alpelisib therapy. Int J Biol Sci. 2025;21(6):2462-2475. Published 2025 Mar 19.

[4]. Kim KJ, et al. PI3K-targeting strategy using alpelisib to enhance the antitumor effect of paclitaxel in human gastric cancer. Sci Rep. 2020;10(1):12308. Published 2020 Jul 23.